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EXTENSION LECTURES

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THE DISEASES AND TREATMENT OF THE INVESTING TISSUES OF THE TEETH

BY

ARTHUR D. BLACK, A.M., M.D., D.D.S.

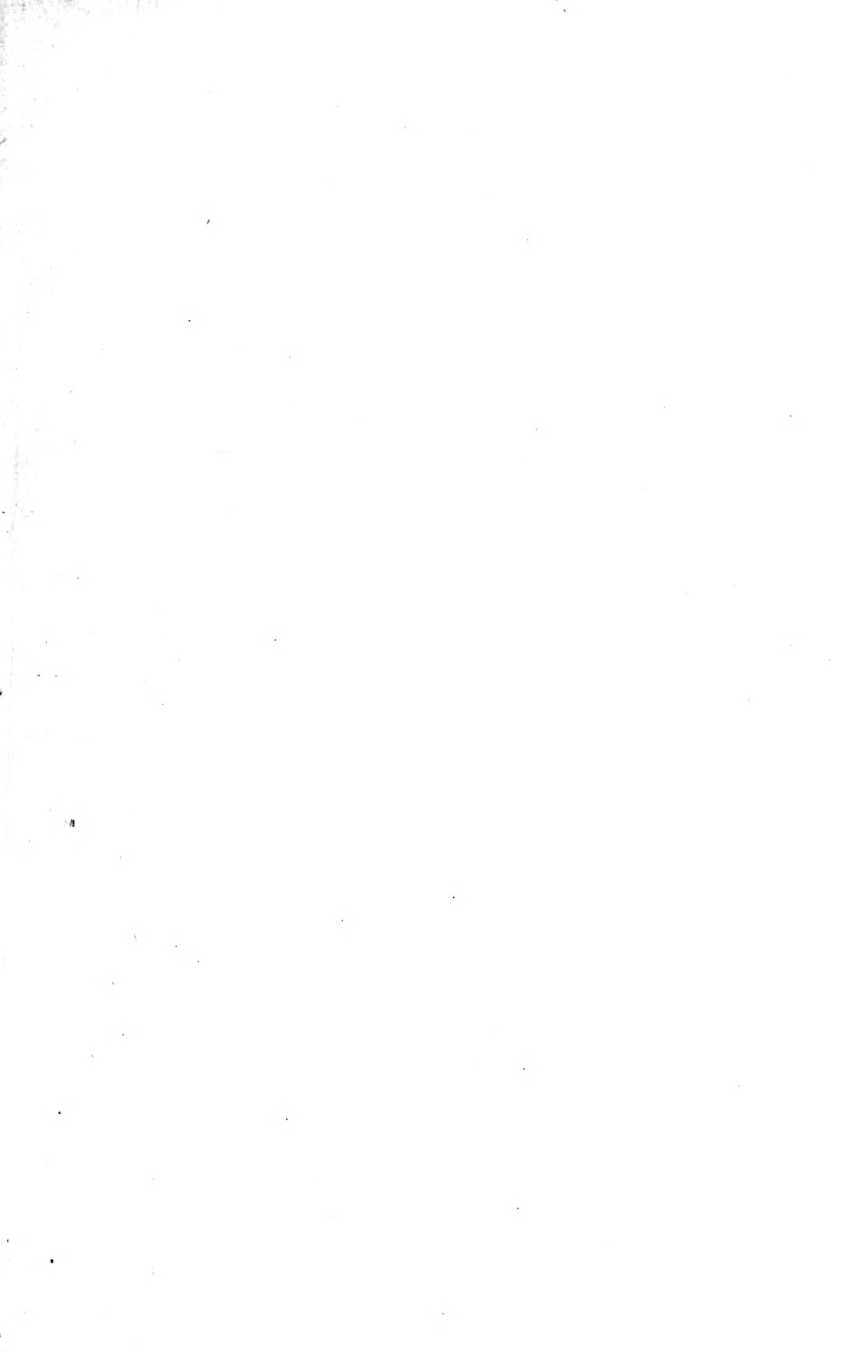
Professor of Operative Dentistry and Special Dental Pathology, Northwestern University
Dental School, Chicago.

DECEMBER 27, 1915—JANUARY 7, 1916

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INTRODUCTION*

Before taking up the work for which we are assembled, I wish to express my appreciation of the honor conferred by the University of California in extending to me an invitation to be one of the lecturers of this Institute. I am pleased also to say a few words in commendation of the action of the University of California in adding dental courses to its extension work. It is a compliment to the profession that the importance of our service to humanity is thus recognized, and I trust that these courses will in future years continue to receive the hearty support of the profession. I wish, before this class, to compliment Dean Millberry for having seen the need of courses under University control, by men without the sphere of influence of commercial interests, and for having suggested the plan to the University authorities. Such courses should stimulate the members of the profession to keep closely abreast of the times and improve the general average of dental service. Thus we see that the University of California is promoting a scheme in which the people of the entire State should be interested, for they will in the end be the beneficiaries.

The treatment of disease improves with more complete knowledge of pathology. For each disease, we should be able to apply treatment closer to the source, more directly to the cause, or actually to prevent it, as our understanding of the pathology becomes more perfect. We are all familiar with the gradual advancement in the treatment of typhoid fever, especially in the prevention of it by improved sanitation when the methods of transmission became known, and by vaccination after the effect of the infection in the development of antibodies was understood. The discovery of the role which the mosquito played in the transmission of yellow fever and malaria made it possible for our government to carry to completion the construction of the Panama Canal, the failure of the French attempt having been due principally to the ravages of these diseases.

A careful review of the literature of dentistry reveals little other than very meager statements of the pathology of the diseases of the peridental membrane. The very large majority of writings on this subject concern themselves with names applied by various authors, with theories as to local and systemic causes, and with statements emphasizing the necessity of removing deposits from roots of the teeth. Very few men seem to have made a serious effort to study

* This course of lectures follows closely the recently published book *Special Dental Pathology*, by the late Dr. G. V. Black (Medico-Dental Publishing Co., Chicago; Claudius Ash, Sons and Co., London, 1915), credit to which is hereby acknowledged. The illustrations herein were reproduced from the same book, as were about one hundred and fifty stereopticon slides used to illustrate the lectures.

and present in detail the pathological changes which take place or to differentiate the various diseases to which the investing tissues are subject. It is not surprising, therefore, that the many plans of treatment have generally been unsuccessful. As a basis for the institution of more rational treatment, this course of lectures will be devoted largely to studies of the histology, physical functions and pathological changes in these tissues. After a time we will come to realize that many of us have been endeavoring to accomplish the impossible.

The peridental membrane is defined as the soft tissue which serves to connect the root of the tooth with the bone of the alveolar process. We might refer to it as the connecting link between chronic infection of the mouth and the general health, since there is practically always a break in this tissue which permits the infective agent to enter the circulation and be carried to distant parts. We might also think of this tissue as that which must bind together the medical and dental professions in their fight against the ever-increasing list of diseases which are recognized as occurring secondary to focal infections. We are concerned chiefly with the dentist's part in protecting the health of his patients, but we will not overlook his duty to conserve the teeth.

Disease of the peridental membrane does not occur except as a result of (1) a preceding gingivitis or (2) the death of the dental pulp. In the one case there may occur a detachment of the tissue from the cementum beginning at the gingival line of the tooth, with the formation of a pus pocket alongside the root; in the other, the detachment occurs about the apex of the root with the development of a chronic alveolar abscess. It will be noted later that the pathological changes in both cases are similar and that we are confronted with the same difficulties in protecting the general health from the effects of these foci. We will come to realize also that the danger to health demands the elimination of all such foci.

The chronicity of these diseases has led to much confusion in our ideas and knowledge of the pathology. It is not unusual for cases to run twenty or thirty years before all of the teeth are lost, and few dentists have the opportunity to observe many cases from beginning to end. It is not strange, therefore, that we have failed to associate the early symptoms with those of the well-established lesion. Especially have we failed to recognize the relationship between the apparently harmless gingivitis and the secondary serious and generally incurable pericementitis. We must learn to correlate the clinical pictures presented by many cases in various stages of progress and, from these, make a composite which will give us a better understanding of the progressive changes which occur.

We must differentiate several distinct diseases of the gingivae and peridental membrane as to their causation, pathology, and treatment, and

in doing this we must have a new nomenclature. Terms which have been applied to a group of diseases must be dropped and others substituted which designate each condition; for the retention of the general terms serves to continue in our minds the confusion which has prevailed. In order to have a clear understanding of the pathology, we require terms which will separate definitely the inflammations of the gingivae from those of the peridental membrane, for it will be pointed out that we may generally cure the one, and practically never the other. Therefore, successful preventive treatment of diseases of the peridental membrane necessitates a clear understanding of the pathology, a recognition of the early stages, and the institution of treatment while there is opportunity to effect a cure.

FIRST LECTURE

HISTOLOGY AND PHYSICAL FUNCTIONS OF THE INVESTING TISSUES OF THE TEETH

The gums, gingivae, cementum, peridental membrane, and the bone of the alveolar process may all be considered as the investing tissues of the teeth. It is important that we have a clear understanding of the histological structure and physical functions of these tissues, in order that we may appreciate the significance of the pathological changes which occur. This will, in turn, lead to more rational treatment. It is especially desirable that we realize the interdependence of these structures upon each other in the performance of their normal functions.

GUMS AND GINGIVAE

The gums and gingivae are made up of a pavement epithelium supported by a base of connective tissue. The epithelium of the gingivae is more dense than that of the gums, the cells being very flat and closely packed together on the surface. They are well supported by many long legs of connective tissues, each of which carries one or more blood vessels far into the epithelial covering. Provision is thus made for the rapid regeneration of the superficial cells, which are frequently injured or worn away as a result of the wear and tear to which they are subjected in mastication. This arrangement also insures prompt healing of injuries to the gingivae and enables this tissue to withstand long-continued or often repeated irritation without serious harm. The gum tissue is a rather insensitive tissue, offering little complaint to injuries which would excite much pain in other tissues. Occasionally, however, long-continued irritation, as the rubbing of the edge of a denture, will develop extreme hypersensitiveness.

It will be noted from the above that one of the important functions of the gingivae is that of protecting the underlying peridental membrane and alveolar process. In fact, this seems to be the principal function of the gingivae. In addition, they doubtless are of material service in maintaining the teeth in the line of the arch.

It is important that we be familiar with the nomenclature of the gingivae and peridental membrane. We may apply the term gingivae to all the soft tissue which rests upon and extends crownwise from the

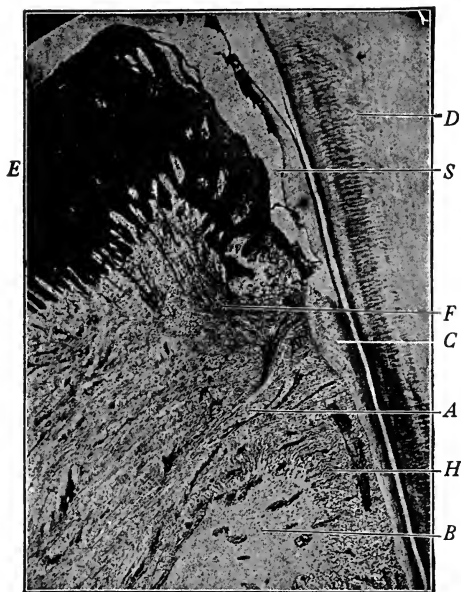


Fig. 1. Longitudinal section through the gingiva and the gingival portion of the peridental membrane, (E) Epithelium. (D) Dentin. (C) Cementum. (S) Subgingival space. (F) Free gingivae group of fibres. (A) Alveolar crest group of fibres. (H) Horizontal group of fibres. (B) Bone of alveolar process.—*Noyes*.

crest of the alveolar process. This may be subdivided into the body, which, encircling each tooth, extends from the crest of the alveolar process to the level of the gingival line of the tooth, the gingival line being the line of junction of cementum and enamel. The free gingivae are those portions which overlie the enamel on the buccal, labial and lingual surfaces of the teeth, and the septal gingivae are the similar portions which occupy the septal or interproximal spaces. We will have occasion to refer frequently to the subgingival spaces—those spaces between the free gingivae and the enamel, or between the septal gingivae

and the enamel. Under normal conditions, a thin, flat blade may be passed into the subgingival space, between the free gingivae and the enamel, until it comes in contact with the attachment of the periodontal membrane to the cementum at the gingival line of the tooth.

The gingivae and periodontal membrane contain several groups of principal fibres which may be named and briefly described in order, beginning at the gingival line and progressing toward the apex of the root as follows:

The free gingivae group is composed of those fibres which extend outward from the cementum just beyond the gingival line and then turn

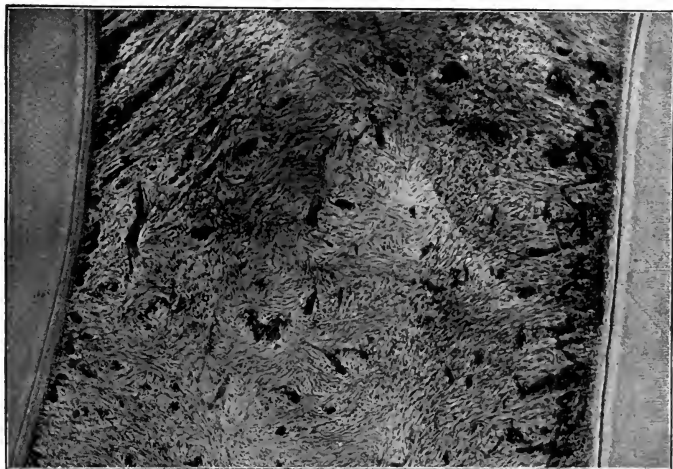


Fig. 2. A portion of the periodontal membrane between two incisors of a young sheep, showing the trans-septal fibres extending from tooth to tooth.—Noyes.

occlusally into the free gingivae. These fibres help to support the free gingivae and assist in maintaining their close adaptation to the teeth.

The trans-septal group consists of a strong band of fibres passing from tooth to tooth through the body of the septal gingivae, occlusally of the crest of the interproximal portion of the alveolar process. The principal function of this group of fibres is to maintain the contacts between the various teeth—to keep the contacts tight. It is necessary to appreciate the function of these fibres in order to understand the movements of the teeth and the progressive involvement of several proximal surfaces from an original pus pocket on a proximal surface. This will be fully explained later.

The alveolar crest group consists of those fibres which extend outward from the cementum and are attached to the crest of the alveolar process. Their principal function seems to be to steady the tooth against lateral strain.

The horizontal group consists of those fibres which extend outward at right angles to the long axis of the tooth and are attached to the bone of the alveolar process, just below its crest. These fibres act with those of the alveolar crest group in preventing too much lateral movement of the teeth.

The oblique group consists of the fibres which make up the bulk of the periodontal membrane. They are attached to the greater part of the surface of the root, and extend in an oblique direction occlusally to the bone of the alveolar process, serving to swing the tooth in its socket and support it against the stress of mastication.

The apical fan-shaped group consists of the many bundles of fibres attached about the apex of the root which radiate in all directions and are attached to the surrounding bone. These fibres tend to maintain the apex of the root in its central position in the socket.

CEMENTUM

The cementum is one of the most important tissues to be considered, for its peculiar structure is in large measure responsible for the chronicity of diseases of the periodontal membrane. The cementum is continuous growing, being gradually built by the cementoblasts which lie upon its surface. These cells are to be considered as an integral part of the periodontal membrane, as they occupy the space between the fibres and, by their action, build cementum around the fibres, thus attaching them to the root.

Cementum is closely analagous to the subperiosteal bone. It is built by cementoblasts lying on its surface, as is subperiosteal bone by the osteoblasts on its surface. The most important difference is that bone has a circulation of blood within and throughout its structure, while cementum has not. This is a very important fact to remember. When there is a suppurative detachment of the periosteum from bone, the underlying bone dies. Then, as a result of the circulation within the bone, a line of demarkation is established and the dead bone is exfoliated. When the periodontal membrane is detached from the cementum by suppuration, the death of the cementum occurs in the same manner, but owing to the lack of circulation within the cementum, the dead portion cannot be thrown off, but remains as a constant irritant to the overlying tissue. This is the principal factor in maintaining the chronicity of the pus pocket alongside the root and the chronic alveolar abscess.

PERIDONTAL MEMBRANE

The peridental membrane contains many specialized elements. The various groups of principal fibres have already been mentioned, as have the cementoblasts and osteoblasts. There are also many strings of epithelial cells which form a more or less definite network about the root of the tooth. The function of these cells has not been definitely determined. Some investigators think they are the remains of those cells which formed the enamel organ and are liable to break down easily as a result of certain toxins in the circulation. Others contend that they have been so placed for the especial purpose of combating infections of the peridental membrane—that they multiply rapidly and accomplish the encystment and destruction of the infective agents and their products. We are inclined to the latter view.

The blood-supply of the peridental membrane is unusually good. Vessels enter about the apex of the tooth and pass alongside the root, other vessels enter over the crest of the bony alveolus, and still others pass directly through the bone. With this triple source of supply the practice of removing the pulps of teeth with the thought of diverting an increase of blood to the peridental membrane would seem to be an unwarranted procedure.

ALVEOLAR PROCESS

The alveolar process is true bone. However, it should be considered an appendage of the tooth. It is built about the tooth as the tooth grows; it disappears following the extraction of the tooth. It seems logical to state that the physiological process of absorption, by which the peridental membrane and alveolar process are removed following the extraction of a tooth, also plays a considerable part in the changes which take place following suppurative detachments of the peridental membrane from the cementum. We should expect the detached fibres, as well as the bone to which their outer ends are attached, to be absorbed, even though they were not involved in the suppuration.

It seems, therefore, that there is a physiological interdependence of the various investing tissues which requires that the health of all be maintained; that serious injury to one is likely to impair them all; and that the gingivae stand guard to protect these underlying tissues. As we study the diseases of these tissues we will come to appreciate more and more the importance of preserving the gingivae in good health in order to prevent disease of the peridental membrane.

SECOND LECTURE

STUDIES OF SALIVARY CALCULUS INFLAMMATIONS CAUSED BY SALIVARY CALCULUS AND TREATMENT

In view of the fact that the injuries due to deposits of salivary calculus have been recognized since the earliest historical times, it seems strange that so little effort has been made to study the causes and nature of the deposit. Credit is due Dr. Henry H. Buchard for having made the first serious effort in this direction.* Dr. Buchard's theory was based on the fact that water containing carbon dioxide will dissolve a greater quantity of various salts than it will without the gas, and its ability of dissolving these salts is further increased by the addition of more of the gas under pressure. If water is charged with carbon dioxide under pressure in a closed tank, its power of dissolving the salts will be increased in proportion to the quantity of the gas. Then, if the pressure is released, the excess of gas will be given off and the corresponding excess of salts held in solution will be precipitated. In the human body, the fluids are charged with carbon dioxide under the blood pressure, and are therefore capable of holding excessive amounts of calcium and other salts in solution. When the fluids are discharged (as the flowing of the saliva from Stenson's duct), they are released from the blood pressure, and the excess of carbon dioxide escapes, while a due proportion of salts is precipitated. It was reasoned that in the mouth this precipitate would be caught in a curd formed by the action of lactic acid on the mucus, the lactic acid being formed by micro-organisms normal to the mouth, and the precipitated salts and the curd would settle down in out-of-the-way places and by additions would finally form a hard mass of calculus.

It was not easy to prove this theory. Dr. Burchard, in his experiments, found that saliva collected in a test-tube, and allowed to stand, would be cloudy within twenty-four hours and it was thought that this cloud was the precipitate of calcium salts.

This was the generally accepted theory until Dr. G. V. Black undertook his long series of experiments about seven years ago. He first duplicated Dr Burchard's experiments with the same results. It then occurred to him that it was not proven that the cloud in the test-tube

* *Dental Cosmos*, vol. 37, 1895, p. 821.

was calcium salts. He contrived to centrifuge the contents of the tube, collecting it on a microscopic cover-glass held in the bottom of the tube, and found that the cloud was composed entirely of a growth of micro-organisms.

Many new lines of investigations were then taken up. Our time is too limited to do more than recite certain of the more important findings. Systematic examinations were made of artificial dentures and it was noted that the mucus, which gives a denture a greasy feeling, could be readily washed off with the water running from the hydrant, but that there would frequently remain a new soft deposit of calculus. It was observed that this could be easily removed with a brush and plain water if this was done before the deposit had remained as long as fifteen or twenty hours, but that it was difficult or sometimes impossible to remove the deposit with the brush if it remained twenty-four hours. This, of itself, offers a most practical suggestion for the prevention of the accumulation of these deposits on the teeth by thorough brushing twice daily.

An upper denture was then constructed with a little rectangular gold frame attached to the vulcanite above the buccal surfaces of the molars. This frame was held in place by two screws and was so arranged that a specially ground cover-glass was held under the frame and could be removed and carried to the microscope for examination without disturbing whatever might be collected upon it. It was thus possible to secure and examine deposits in all stages, from the very fresh soft deposits to those which were quite hard. Various staining methods were employed to bring out more clearly the structural characteristics. It was also possible to make photomicrographs of all specimens.

This line of investigation developed several important facts. It was quickly recognized that the deposit was not a precipitate, as had been supposed, but that the forms were all spherical, and the material constituting the deposit was a calco-globulin, and this globulin brings with it the calcium elements which eventually, with the decomposition of the globulin, form the hard deposits. This globulin is called the agglutinin of salivary calculus. Repeated examinations showed the primary deposit to consist of minute spherules (smaller than red blood corpuscles) which gradually coalesced to form larger masses of more or less rounded forms.

It was also determined that the outpouring of the deposit is decidedly paroxysmal; this is one of the striking characteristics. It was later discovered that the paroxysms are of comparatively short duration and occur within a few hours after a heavy meal. If, for example, a heavy meal were eaten at noon, the cover-glass on the denture would remain clear until possibly two o'clock, when the paroxysm of deposit would

begin. It would continue for an hour and a half or two hours and then cease. A new glass placed at this time would remain clear.

These experiments seem to have established the fact that a heavy meal, especially when well digested, puts into the system more of nutrient material than is required, and that a proper balance is restored by the paroxysmal outpouring of the caleo-globulin with all the secretions and excretions, each of the body juices and fluids having its due proportion. Persons subject to frequent heavy deposits were able to continue for weeks without any deposits when they reduced the amount of food taken a little below that to which they were accustomed. During such a period, a single heavy meal would result in a paroxysm of deposit. It seems to make no difference what foods are selected for the heavy meal.

Many other experiments were performed which can not be enumerated in this course. One other thing should be mentioned—a special grinding machine was constructed and a technic developed by which very thin sections of the hard salivary and serumal deposits could be ground thin enough to be available for microscopic study and photographing. These showed the structure of the hard deposits to be similar to the soft deposits caught on the cover-glasses.

INFLAMMATIONS DUE TO DEPOSITS OF SALIVARY CALCULUS

Deposits of calculus always occur on hard substances. In the mouth we find these deposits on the teeth, and on crowns, bridges, artificial dentures, and other appliances. The position of the deposit is most likely to be near the orifices of the ducts from the salivary glands; upon the buccal surfaces of the molars and the lingual surfaces of the lower incisors. The initial deposit is usually at the position of the crest of the free gingivae and occurs because there has been a slight blunting of the normally thin edge. Such a blunting, or some irregularity of form, seems necessary for the initial deposits to become attached. This fact should emphasize the importance of keeping the gingivae in good health with normal thin crests.

GINGIVITIS CAUSED BY DEPOSITS OF SALIVARY CALCULUS

When the initial deposit in any position has become hard, there is an irritation of the underlying gingivae. This form of gingivitis, with the gradual accumulation of the deposit, is distinct from others. There are frequent suppurations of the shelf of gingivae which is in contact with the deposit. As the deposit increases, it gradually destroys the gingivae and is apt to replace in contour the lost tissue. The most striking feature of this, in comparison with certain other forms of gingivitis, is that there is little tendency for the inflammation to involve any other tissue than that of the shelf which is in contact with the deposit.

PERICEMENTIS CAUSED BY DEPOSITS OF SALIVARY CALCULUS

If the deposit is not removed, it is likely to destroy all the gingivae and encroach upon the periodontal membrane, gradually destroying not only the periodontal membrane, but also the bone of the alveolar process and overlying gum tissue. In all of this there is the tendency for the attachment of fibres of the periodontal membrane to be maintained to the level of the tissue destroyed by the deposit. There is little tendency to the stripping off of the periodontal membrane from the cementum and the formation of pus pockets alongside the roots. Deposits of salivary

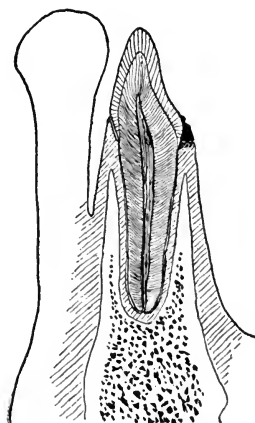


FIG. 3

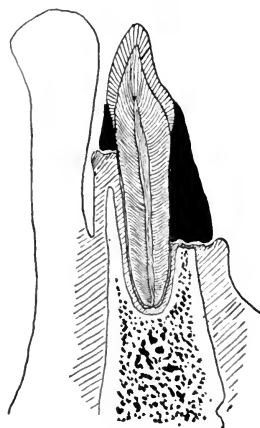


FIG. 4

Fig. 3. Drawing illustrating a slight deposit on the lingual surface of a lower incisor which has caused a gingivitis only, not having progressed far enough to involve the attachment of the periodontal membrane to the cementum.

Fig. 4. Drawing illustrating a still greater destruction, including also the labial tissues.

calculus should not therefore be considered a cause of pus pocket formation, as has commonly been thought.

As the destruction of the investing tissues continues, the patient may complain of occasional pain and soreness and the teeth may gradually become loose, so that eventually they are lost. This is a slow process, usually extending over many years, and as suppurations are frequent, the menace to the health is probably greater than has been realized. The thin-walled blood vessels in the granulating surfaces offer micro-organisms easy access to the blood stream.

TREATMENT

The treatment of the inflammations caused by deposits of salivary calculus should always be undertaken with the possibilities of prevention well in mind. It has been stated that fresh deposits are so soft for fifteen or twenty hours that they may be easily removed with a brush and plain water. Therefore, every patient who may be induced to brush the teeth thoroughly twice every day can prevent the inflammation and destruction caused by the hard deposit. This has been sufficiently well demonstrated in many cases to leave no room for doubt of its efficacy.

Our management of cases presenting with deposits should be with the view of gaining the co-operation of the patient to the end that future deposits will be prevented. The removal of the deposits is not difficult. They are practically always in sight; not covered by the gingivae. Only a few simple instruments are required. After the removal of the deposits, the surfaces of the teeth should be polished. If there is much inflammation, frequent rinsing of the mouth with warm salt solution will keep the field clear and lessen the patient's discomfort.

In such cases it is usually best not to advise the patient as to the technic of proper mouth hygiene at the time when the deposits are removed. This should be reserved until the inflammation has subsided. The patient should be given another appointment and should then receive whatever instruction seems necessary to gain his interest and co-operation. This should include a few brief statements explaining the nature of the fresh deposit and the possibility of preventing its accumulation by faithful brushing twice daily. It should also include mention of the danger to the general health, as well as the probable eventual loss of the teeth. Brushes should be selected, their movements demonstrated and instructions given to the finest detail.

If possible, a definite arrangement should be made for a subsequent examination, so that we may know how well the home care is progressing. A chart of the mouth, showing locations of deposits, should have been made at the time of examination and each position should be carefully inspected. deposits removed if there are any, and additional instruction given the patient. Considerable diplomacy is necessary in the management of many patients, but one who comes to realize fully the effectiveness of this plan is sure to become more and more enthusiastic and more successful in gaining and holding the interest and co-operation of the patients.

THIRD LECTURE

GINGIVITIS CAUSED BY DEPOSITS OF SERUMAL CALCULUS WITH TREATMENT

In considering the inflammations of the gingivae caused by deposits of serumal calculus, we have to do only with the deposits which occur on the enamel of the subgingival spaces. A serum is normally poured out into these spaces to keep them moist. We should expect this serum to contain its proportionate share of calco-globulin, whenever there is a paroxysm. As the quantity of serum is small compared with the amount

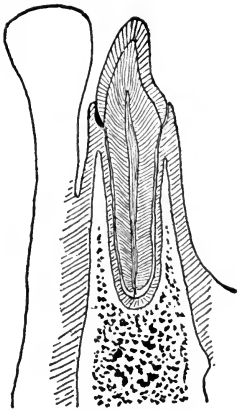


FIG. 5.

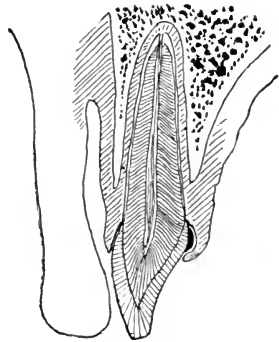


FIG. 6.

Fig. 5. Drawing illustrating a deposit of serumal calculus under the free gingiva on the labial surface of the enamel of a lower incisor tooth.

Fig. 6. Drawing illustrating a similar deposit on the lingual surface of an upper incisor. Suppuration of the periodontal membrane, resulting from deposits in this position, causes the teeth to move labially, and such cases are generally hopeless, after much progress has been made.

of the saliva, so will the deposits be small and slow of formation. The causes are the same, and the nature of the deposit is the same as salivary calculus. We will often find both deposits in the same mouth. However, proper care or vigorous mastication may prevent the accumulation of salivary deposits, while serumal deposits are being laid down.

Owing to the fact that the gingivae naturally hug closely about the teeth, the deposits occurring in the subgingival spaces are likely to be

compressed while soft, and harden as flattened scales. If the close adaptation of the gingivae is lost as a result of inflammation or detachment of the peridental membrane, the form of the deposit will generally be nodular instead of flat.

Clinically, these deposits may be divided into two groups: one in which there is a general deposit in the subgingival spaces in the form of narrow bands more or less completely encircling the teeth. The inflammation of the overlying gingivae may or may not be apparent. In such mouths deposits of salivary calculus are usually present. In the other group, the deposits will be confined to certain teeth, others being free. In such cases an inflammation of the gingivae about the particular teeth has usually preceded the occurrence of the deposit. An irritation, such as the wedging of food through an open contact, or an ill-fitting crown, causes the outpouring of greatly increased amounts of serum from the inflamed gingivae, thus delivering to these spaces a correspondingly increased amount of calco-globulin for deposit. We therefore find considerable deposits in such locations, with little or none elsewhere in the mouth. It is not uncommon to find deposits on the lingual surfaces of the upper incisors, from the irritation caused by food forced against the gingivae by the lower incisors, especially if there is much overbite.

In all cases in which there are deposits of serumal calculus in the subgingival spaces, these cause inflammation of the overlying gingivae, and occasionally suppuration occurs. The tissues may recover without serious harm having been done. Sooner or later, however, a suppuration will involve the attachment of the peridental membrane at the gingival line and a little pus pocket will be formed. In this way these deposits become the real exciting cause of suppurative detachments of the peridental membrane in probably twenty per cent of cases.

TREATMENT

The treatment of inflammations caused by these deposits should be along the same general lines as that mentioned for salivary deposits. More delicate instruments are required, also a higher degree of finger skill. One must learn to feel the deposits and to recognize a surface free of deposit in positions where he cannot see. While proper brushing by the patient will assist in keeping the gingivae in good health, the use of a rubber bulb syringe to wash out the subgingival spaces thoroughly twice a day is much more efficacious. This requires more careful training and instruction and more of enthusiasm to get patients keyed up to the point at which their interest and care will be maintained. A similar plan of recording positions of deposit, of subsequent examinations, etc., should be followed. Even in mouths free from these deposits, nothing



Figs. 7, 8, 9. Three molar teeth showing "rings" of deposits of ser-umal calculus on the enamel of the subgingival space. Frequently these "rings" encircle the crown. Specimens from Northwestern University Dental Museum.

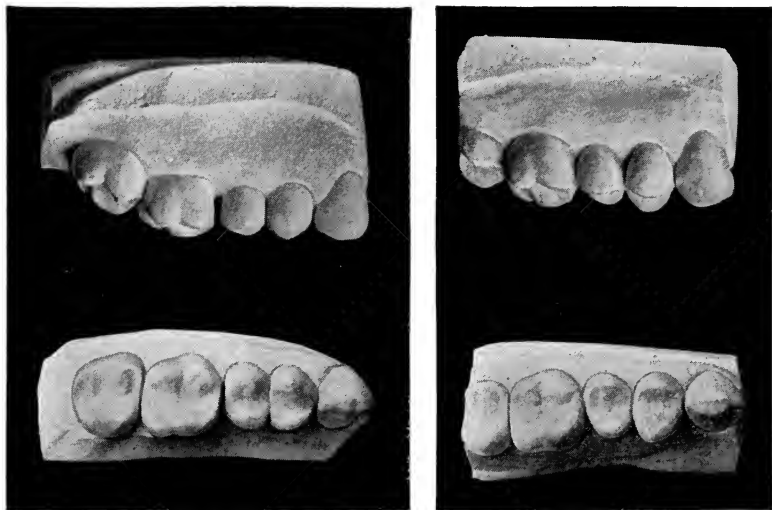


Fig. 10. The position of the rubber bulb syringe in washing the subgingival spaces or pus pockets. The end of the nozzle should touch the enamel of the tooth near the crest of the gingivae as it is passed along the arch, the angle being such that the water or solution will be forced into the subgingival spaces. This is the most effective means of preventing deposits of ser-umal calculus.

will contribute so greatly to the maintenance of the gingivae in good health as the twice daily irrigation of the subgingival spaces with warm water or normal salt solution.

GINGIVITIS CAUSED BY INJURIES, WITH TREATMENT

In our studies of the histology and physical powers of the investing tissues, the protective function of the gingivae was noted. The gingivae are naturally equipped to withstand severe punishment, which they do in a remarkable way. The fact that they will maintain themselves so long under continued irritation, together with our failure to recognize these injuries as forerunners of more serious lesions of the periodontal



Figs. 11, 12. Photographs of plaster models of a case before and after contact restoration. The patient presented with a slight pocket on the mesial surface of the root of the first molar on account of the open contact. The mesial surface of the first molar and distal of the second bicuspid were free from decay and had not been filled. The separation had occurred as a result of flat fillings in the mesial of the second bicuspid and distal of the first bicuspid. These fillings were removed, and a Perry separator was applied on several occasions to move the second bicuspid back into contact with the first molar, it being held there for a time with fillings of base-plate gutta-percha. Later, permanent fillings were made, restoring normal conditions, as shown in Fig. 12. It was necessary to relieve the occlusion on the distal slopes of the cusps of the second bicuspid as it was moved.

membrane, has led to much abuse and neglect. Yet it seems fair to state, from statistics which will be presented, that possibly seventy-five per cent of the pus pockets alongside roots of the teeth are the direct result of long-continued and neglected injuries to the gingivae.

With an irritation of the gingivae from any form of injury, there is opportunity for suppuration, with subsequent involvement of the attachment of the periodontal membrane. As has been mentioned, the irritation is likely to result in a deposit of serumal calculus, which becomes a secondary cause of the further progress of the case. In connection with the more acute suppurations there is often considerable pain. Absorptions of the gingivae occur as a result of the constant irritations and repeated suppurations.

It would be almost impossible to cite all the causes of these injuries. Lack of contact of the teeth due to extractions of neighboring teeth, flat fillings, crowns, etc., improper contact of teeth, irregularities of contour due to sharp edges of cavities, bad margins of fillings and crowns, etc., abuse of these tissues in operations, and injuries by patients in cleaning, are the most frequent causes.

In cases in which contacts are not normal, and food is wedged between the teeth, the septal gingivae become inflamed from the repeated impactions of food, also from the efforts to remove it. These impactions will, after a time, result in the absorption of the central portion of the septal tissue, while the buccal and lingual portions may be pushed outward in their respective embrasures. Their appearance is that of slightly swollen festoons. Later on, as the central portion is further depressed, there is likely to be some absorption of the buccal and lingual portions also. This may continue until the absorption has included much of the bone of the alveolar septum, with deep pus pockets on the proximal surfaces of the teeth.

Some patients will complain bitterly of the pain caused by slight impactions of food, others will seem unconscious of the presence of considerable accumulations in many spaces. Treatment should therefore be undertaken on the basis of the impaction and inflammation, rather than on the complaint of the patient.

Several years ago, in order to get some reasonably reliable data as to the frequency of the various forms of gingivitis, I requested a number of dentists in various sections of the country to assist me in collecting certain statistics. I sent a number of examination cards to each man with a request that he make a careful record of the areas of gingivitis found in the mouths of young adults, who had no disease of the periodontal membrane. I present herewith a brief summary of the results of the examination of 500 mouths of persons between twenty and thirty-five years of age.

Of the 500 mouths examined, but 25 were reported as having no gingivitis. For the other 475 persons, 4265 areas of gingivitis were reported—an average of 8.53 areas per person for the entire 500. Of these areas, 1348 were due to deposits of salivary calculus. These were in the mouths of 39 per cent of persons examined, and represent 31 per cent of all areas of gingivitis. In recording these, each surface of each tooth having a deposit was counted as one area. For example, a deposit on the lingual surface of the lower incisors and cuspids was counted as six areas.

Five hundred and sixty-three areas were reported as having deposits of serumal calculus on the enamel of the subgingival spaces. These were in the mouths of 15 per cent of persons examined, and represent 13 per cent of all areas of gingivitis. Many who had deposits of salivary calculus also had serumal deposits; 140 persons were reported as having either or both, leaving 360 without deposits of either kind.

There were 2364 areas due to other causes than deposits. Of these, 783 were due to bad margins of fillings or crowns, 496 to lack of contact of fillings or crowns, 305 to improper contact of fillings or crowns, 263 to malpositions or atypical forms of proximal surfaces, 255 to lack of contact with no decay of proximal surfaces, 233 to caries, and 19 to worn contacts.

If we add together the number of areas due to bad margins, lack of contact or improper contact of fillings or crowns, the total is 1584. This number of areas, 37 per cent of all, may be properly charged to imperfect dental operations. This is an average of more than three such areas per mouth, and would seem to indicate that fully one-third of the pus pockets are due to lack of care in operative and prosthetic service.

There was a time when no consideration whatever was given to the soft tissues in the performance of either filling or crowning operations. When files were used to separate the teeth, and wedges were driven between them, the importance of preserving the investing tissues could not have been appreciated. It has only been within comparatively recent years that the attitude of the profession has begun to change. Not until we realize the direct relationship of these areas of gingivitis to the more serious detachments of the periodontal membrane which follow, will we be as careful as we should be in the finer technic of all operations so that the number of these injuries will be reduced to the minimum.

TREATMENT

In the treatment of cases of this kind, it is essential that we should first make careful search for the cause. When this has been found, the treatment will be indicated. If, because of an open contact, food is

wedging between the teeth and injuring the septal gingivae, we must, if possible, learn the cause of the open contact. It may be that the cusp of a tooth in the opposite arch needs a little grinding, or the extraction of a tooth may have permitted movement of others, opening the contacts. Many times it is a flat filling which did not restore contact, or it may be any one of many things. Most of these are easily corrected and the gingivitis will promptly subside when the cause of the irritation is eliminated. It is not so much the difficulty of correcting these conditions, as it is the necessity of appreciating the importance of it.

When a contact is too broad, though tight, it should be corrected by trimming to proper form. Other things should be done as required in particular cases. Many require careful study. Close attention to these things will impress the possibilities of prevention by greater care in every operation performed.

In all of the operations for contact restoration, one of the most important things is proper separation of the teeth without pain or injury to the soft tissues. Notwithstanding all of the more recent devices, I know of nothing so satisfactory as the Perry separators. These seem to meet every requirement. A little experience is necessary to be able to adjust them properly, and they are more expensive than other devices, yet in the long run they are very economical, when one considers the time saved and the benefits gained by their use.

FOURTH LECTURE

CHRONIC SUPPURATIVE PERICEMENTITIS

The term "chronic suppurative pericementitis" is applied to that disease, the essential characteristic of which is the formation of a pus pocket alongside the root of a tooth. This term was selected because it is closely descriptive of the condition. One of the marked features of this disease is its chronicity, to which reference has already been made. It is also essentially a suppurative disease and it is notable that the suppurative process strips the soft tissue from the cementum, which makes the word *pericementitis* especially applicable. There is room for question in many cases, whether or not the bone of the alveolar process is involved directly by the suppuration, or is absorbed secondarily by purely physiological processes. Therefore, terms which indicate an inflammation of the alveolar process do not place the principal tissue involvement where it really occurs. Whether this term, or some other, comes to be finally accepted and used, it is imperative that a name be found which will apply to this condition as a pathologic process different from the others to which the investing tissues are subject.

The local causes leading to the formation of pus pockets have been reviewed. We have said that a gingivitis always precedes the pericementitis, and have emphasized the fact that deposits of serumal calculus and injuries to the gingivae are the principal local exciting causes. In doing this we do not overlook the effect of the systemic condition



Figs. 13, 14. Panoramic radiographic views of the upper and lower jaws in a case of chronic suppurative pericementitis of long standing. This patient had suffered from gout for five years. The right foot was first swollen and was very painful. In subsequent attacks the ankle was involved. The patient stated that except for this, he had never been sick a day. One lower incisor had become so loose that it was removed with the fingers. Pressure upon the gums caused pus to exude about the necks of many of the teeth. It was advised that all of the teeth be extracted.

in some cases in which local causes are found, and we also appreciate the fact that there are cases in which no local causes are apparent. We are, however, most concerned at this time with conditions which are recognizable and for which definite treatment is indicated.

Many men have searched for a specific organism which might be proven to be the cause of these suppurations. Up to the present time, all such attempts have failed. Careful clinical study of the progress of cases

contraindicates a specific type of infection, as will be explained later.

The recent claim of a number of writers that the endameba is the cause of this disease seems not to be well founded, and the use of emetine has proven a disappointment. It would have been just as logical to select

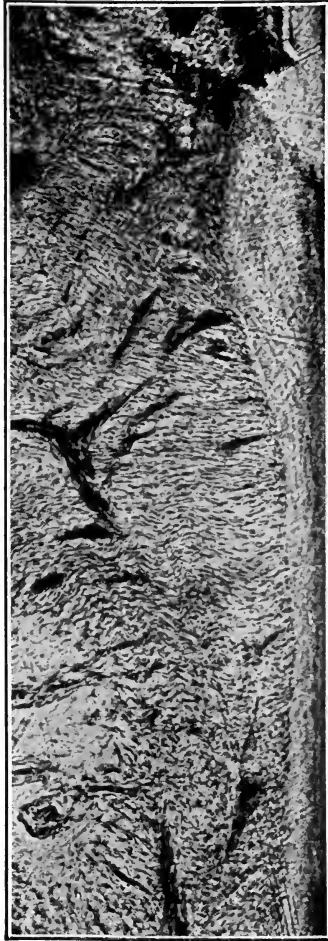


Fig. 15. Normal periodontal membrane. The row of cementoblasts may be seen lying along the surface of the cementum. These cells occupy most of the space between the fibers as the latter enter the cementum. Photograph by Dr. F. B. Noyes.

any one of the other organisms which can be generally found in these pockets, and state that it alone caused this disease.

The symptoms and tissue changes are those of a progressive chronic infection. The appearance of the gingivae may be normal, or the crests may be slightly blunted and swollen, with slight or considerable discoloration. The suppuration occurring because of the inflammation of the gingivae sooner or later involves the peridental membrane and cuts it away from the cementum, beginning at the gingival line. When a detach-

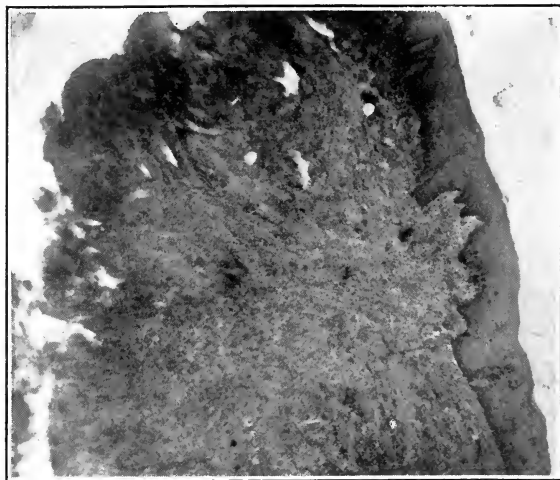


Fig. 16. Section through soft tissue overlying a deep pocket of many years' standing on the labial side of the root of a lower left cuspid; from about the middle of the length of the root. Patient sixty-five years of age. Tissue cut away by Dr. Arthur D. Black on September 29, 1913. Normally the crest of the alveolar process should be present in a section cut in this position. The bone has all disappeared, as have practically all of the fibers of the peridental membrane. Section prepared by Dr. H. A. Potts, photographed by Dr. F. B. Noyes.

ment has been affected, the general tendency is for pockets to slowly progress. This progress is greatest toward the apex of the root, rather than around the root, so that it is not unusual to find very deep, narrow pockets. The cementoblasts are the first of the specialized elements to be destroyed. This seems to occur as a part of the suppurative detachment of the soft tissue from the root. Subsequently, the principal fibres of the peridental membrane gradually disappear from the overlying tissue, and later the bone of the alveolar process to which these fibres were attached disappears also. As has been mentioned, it should be expected

that these tissues would be absorbed following the detachment, regardless of their possible involvement in the suppurative process. The disappearance of the fibres of the peridental membrane may be shown by microscopical examination of tissue cut from these positions; while the absorption of the alveolar process is clearly shown by radiographs.

The soft tissue overlying the denuded cementum presents a granulating surface containing many newly formed, thin-walled blood vessels, which offer favorable opportunity for the many micro-organisms within the mouth to enter the circulation. The cementum itself has necessarily absorbed the products of suppuration and putrefaction, so that a condition could hardly be imagined which would present greater difficulties to a normal re-attachment of the tissue to the root. Every specialized element of the peridental membrane has disappeared, and the tissue which remains lies against a cementum which has been rendered negatively chemotactic by absorbing the products of the suppuration. It is for this reason that these detachments are permanent detachments.

It has been suggested that the outer surface of the cementum should be cut away, with the expectation that an attachment would occur similar to that which takes place when teeth are implanted, transplanted or replanted. We have not time to discuss this point at length at this time, but attention is called to the fact that the attachment in such cases is an unstable one, and it in no sense a re-attachment of the peridental membrane. Where such teeth become firm, the rule is that it is by absorption of the root and building in of the surrounding tissue, a process which, within a few years, results in the loss of the tooth.

Doubtless many conscientious operators have been misled into believing that re-attachment occurs; this has resulted from their failure to observe cases with sufficient care over a long enough period of time. It is the rule that many cases look very much better following treatment, and that there is close adaptation of the soft tissue to the root, so that one may be deceived. If such cases are kept under close observation, it will be found practically always that the pockets are still there, and that there will be a recurrence of the pus formation.

Pain is not a prominent symptom, although the tissues about such teeth will occasionally be very painful when the suppurations are acute, or if the pus penetrates deeply into the surrounding tissues. Most patients will complain that the teeth are periodically sore and that they are raised in their sockets. The greater the progress of the case, the more frequently will pain and soreness be noted.

Deposits of serumal calculus will be found upon the denuded cementum in many cases. These deposits should never be considered a cause, but always a result of the formation of the pocket, as the material for the deposit—the calco-globulin—is brought to the pocket by the serum

escaping from the overlying inflamed tissue. These deposits are frequently nodular, as the overlying tissue does not hug closely about the root, and the deposit is not compressed while soft, as is often the case with the deposit on the enamel. When deposits have occurred on the cementum, they cause additional inflammation of the overlying tissue and in this way contribute to the further progress of the pocket.

The cervical lymphatic glands are occasionally enlarged in advanced cases. There is also, as a rule, an excitation of the salivary glands, requiring the patient to swallow frequently and possibly to drool at night.

Pus pockets may be divided into two groups, according to their location. In many cases the pockets will all be on proximal surfaces, with little or no involvement of buccal, labial or lingual surfaces. In others, all the pockets will be on buccal, labial or lingual surfaces. In the later stages, all surfaces will be involved. It is not uncommon to find a single pocket on the labial or buccal surface, or possibly two or three. The lingual surfaces of the upper incisors are frequently involved as a group.

The tendency is for teeth having pus pockets on one side to move in the direction away from the pocket. This is partly due to the inflammation, but principally to the fact that the balance of pull of the various fibres of the peridental membrane has been disturbed and the fibres on the sound side pull the tooth in that direction. When the pockets are on the lingual side of the upper incisors, these teeth generally move labially and, with the pull of the trans-septal fibres, soon draw the cuspids away from the first bicuspid and open the contacts. The contacts between the incisors and cuspids are also opened by the forward movement, and the septal tissues soon become inflamed from food impactions. When the labial movement of the incisors has once fairly begun, it is seldom checked and after a time the teeth are lost. Very often the forward movement of the cuspids results in the eventual loss of the bicuspid and molars also.

In cases in which the original pocket is alongside a proximal surface in the bicuspid or molar region, the neighboring teeth are likely to become similarly involved on their proximal surfaces, the buccal and lingual tissues remaining intact. When such a pocket occurs, the inflammation within the septal space, together with the pull of the fibres on the opposite sides of both teeth, tend to open the contact between the teeth. When this occurs, there is usually some pressure on next neighboring contacts, with slight movement of the adjacent teeth. With the subsidence of the inflammation, the teeth return to their normal positions. This is repeated again and again, until after a time the frequent movement results in the weakening of neighboring contacts and stringy foods are forced through, causing inflammation of the septal tissues. This continues

until pus pockets are formed on the proximal surfaces of these teeth. In this slow way these cases progress. The movement of the teeth of one arch will often disturb those of the opposite arch and similar inflammations will follow. Thus all the teeth may be lost as a result of a single proximal pus pocket.

Occasionally the pus formed in deep pockets is not discharged alongside the root, but involves the adjacent soft tissues and forms an acute abscess at the side of the root. This is a lateral abscess. It may be mistaken for a true alveolar abscess.

TREATMENT

The key to the treatment of chronic suppurative pericementitis is in the statement that suppurative detachments of the periodental membrane are permanent detachments. With this in mind, we may divide the treatment under three headings: preventive, palliative and radical.

Preventive treatment consists of the carrying out in practice of every measure which will prevent or cure gingivitis and thus protect the periodental membrane. The methods of doing this have already been discussed. This treatment must be by the general practitioner of dentistry and not by a specialist, for it must come to be a part of every dental operation to conserve the health of the investing tissues. This must be done by careful systematic examinations, properly recorded; by the maintenance of good contacts to promote thorough mastication, and careful training of patients in mouth hygiene. This should come to be a considerable part of the practice of each dentist—and it should be the service by which a substantial portion of his income will be earned. Practitioners who follow such a plan will have very few patients in whose mouths pus pockets will occur.

Palliative treatment should be applied in those cases in which pus pockets have formed, but are not bad enough to require the extraction of the teeth involved. It is not possible to give a definite rule by which the line may be sharply drawn between those conditions which demand extraction and those which contra-indicate this operation. More will be said on this point later.

If palliative treatment is undertaken, the first procedure should usually be the removal of deposits from the roots, if deposits are present. It makes little difference what instruments are used, if the operator is able to remove the deposits. A large number of scalers is not necessary, for it is believed that other means should be employed instead of scaling operations if there are deposits in positions of very difficult access. It is certainly of the greatest importance that each operator should develop the best possible finger skill.

The instruments should be sharp and the effort should be made to remove all the deposit and leave the root smooth. In doing this, as much as possible of the cementum should be left on the root. The removal of the cementum causes many teeth to become hypersensitive, so that thermal changes or even the mastication of food is painful. It should be borne in mind that there is no physiological provision for transmission of sensation through cementum. Therefore there should be no sensation transmitted through the pulp in scaling operations, so long as the cementum is intact. Sometimes acid formed by organisms growing within a pocket will soften the cementum, so that it is easily removed, or it may be removed by repeated scaling operations. One case has been presented in which the pulp, within the root-canal, was actually exposed by the too vigorous use of scalers.

If pain is caused in the overlying tissues by scaling operations, novocain may be injected. However, in such cases I have generally preferred to remove the bulk of the deposits, which can usually be done without much pain, at the first sitting, and then, by thoroughly irrigating the pockets with salt solution on two or three successive days, bring about sufficient reduction of the inflammation that the more thorough scaling can be done without causing pain.

Following the scaling, the case should be observed by the dentist until the inflammation has subsided, irrigating all pockets thoroughly at each visit. The patient should then be carefully instructed in the use of the rubber bulb syringe and should be impressed with the necessity of irrigation twice daily with normal salt solution. By this plan the pockets will be kept clean and free from accumulations or micro-organic growths, and the recurrence of deposits will be in large measure prevented. After the roots are once thoroughly cleaned, the irrigation by the patient becomes the most important factor in the prognosis of most cases, and its importance cannot be too strongly impressed. There should of course be an arrangement for subsequent examinations, and further treatment when necessary.

In a limited number of cases, the cleansing operations, and incidentally the scaling, may be simplified by cutting away the overlying soft tissue and thus materially reducing the depth of the pocket. This treatment is most frequently indicated for pockets on labial or buccal surfaces.

Time will not permit a discussion of the reasons for the abandonment of antiseptics in the treatment of these pockets. It need only be said that antiseptics were originally used here for the same reason that they were used in other infected cavities by surgeons, to inhibit the growth of the organisms. Today we should abandon their use for the same reason that surgeons have generally done so, because it has been found that they do more harm than good; that an antiseptic which will be effective against

micro-organisms will also so inhibit the activities of the tissue that nothing is gained. By the use of salt solution, most of the organisms are washed away and the tissues are left in the best condition to destroy the remainder.



Fig. 17. Plaster model of case in which the tissue overlying a pocket on the mesio-buccal root of an upper first molar was cut away to reduce the depth of the pocket and facilitate the cleaning.



Fig. 18. A case in which the distal root of a lower first molar was amputated. The distal half of the crown of the tooth was also cut away, and a gold crown was made to restore the full occlusal surface.

This plan of treatment is quite simple, but is very effective if the co-operation of the patient can be secured. So long as the pockets are kept clean, they are practically well, and both the teeth and the health of the patient are conserved.

Radical treatment for these cases consists of extraction or root amputation. As a general statement, it may be said that we have gone too far in our effort to save these teeth. When so much of the investing

tissue has been destroyed that the tooth is very loose, it should be extracted. If a tooth has deep pockets and is periodically sore enough to interfere with proper mastication, it should be extracted. If the formation of pus can not be controlled, the tooth should be extracted. In most cases, if the disease has progressed to denude the bifurcation of multi-rooted teeth, they should be extracted. Root amputation may be substituted for extraction in cases in which a single root of a multi-rooted tooth is diseased, while the other root or roots are not. The lingual root of the upper first molar offers the best opportunity for successful amputation. It often requires a very careful study of cases to come to a proper decision regarding extraction. We certainly should not be guided by the usefulness of the tooth in mastication alone, as many teeth which are serviceable are a decided menace to health.

FIFTH LECTURE

MANAGEMENT OF CASES SYSTEMIC EFFECTS OF MOUTH INFECTIONS

In the previous lectures, we have discussed the several diseases to which the gingivae and peridental membrane are subject, considering the pathology and treatment of each separately. In many mouths several or all these conditions may be present at the same time. An accurate and complete diagnosis is therefore often difficult, as is the determination of the best plan of treatment. For this reason a definite system of making and recording examinations becomes a matter of first importance. In the clinics accompanying these lectures I have demonstrated a simple plan by which each area of inflammation may be recorded by a number, which will indicate the condition of the tissue, the position, and the cause. It requires a little experience to become familiar with any such plan, yet the advantage gained by having made and recorded such complete surveys of the mouth will fully compensate for the effort. The habit of doing this will sharpen one's observation and put one in position to lay out a comprehensive plan of treatment. Every area of gingivitis, the locations of deposits of both salivary and serumal calculus, the condition of contacts, the location and depth of pockets, teeth which are missing, those which are loose, and many other items are necessary to a complete record. In cases in which there are pockets, radiographs should be made for the additional information which they will give and to verify the instrumental examination. In some cases inquiry should be made into the general physical condition of the patient.

With all the facts obtainable, one is ready to lay plans for treatment. It should first be determined what teeth unquestionably require extraction, then those which should unquestionably receive palliative or preventive treatment. There may be several regarding which there is some question and oftentimes it will be wise to reserve one's judgment regarding these for a little time, possibly for several months, until the attitude of the patient towards the technic of mouth hygiene has been observed. It will be possible to retain, without menace to the health, teeth with tolerably deep pockets in one mouth, if the use of the syringe is faithfully carried out, while in another even quite shallow pockets will constitute a serious menace if they do not receive proper irrigation.

The problem of appearance, and that of replacing lost teeth to provide a means of proper mastication, must have consideration at the same time. Often it will be best to remove some of the "doubtful" teeth in order to give better abutments for a bridge, or if a denture must be made, there may be an advantage in extracting several such teeth.

The general attitude of our people in favor of saving every tooth as long as possible presents one other difficulty in the management of these cases. When it has been determined that certain teeth should be extracted, careful diplomacy must often be used in our advice to the patient. I have, on several occasions, given patients what I am sure was sound advice to the effect that they should have many or all of their teeth extracted, with the result that I did not see them again. We should always bear in mind the fact that the loss of the teeth is a serious thing in the life of most persons, and some time is often required to bring them to a proper realization of the situation which confronts them. It is often best, therefore, first to suggest and later to state definitely what seems best, with our reasons.

The studies by medical men in recent years have brought to us another proposition for very serious consideration; viz., the relation of the local foci of infection to many serious systemic diseases. This relationship has been recognized by a few men for many years, but was not prominently before either the medical or the dental professions until Hunter, of London, wrote his famous article in 1911.

As early as 1891 our own Dr. W. D. Miller wrote as follows: "During the last few years the conviction has grown continually stronger among physicians, as well as dentists, that the human mouth, as a gathering place and incubator of pathogenic germs, performs a significant role in the production of varied disorders of the body, and that if many diseases, whose origin is enveloped in mystery, could be traced to their source, they would be found to have originated in the oral cavity."

Dr. Hunter wrote his first paper in 1900, but it was not until eleven years later, after he had seen hundreds of patients improve or fully

recover from those diseases which are now recognized as occurring secondary to local foci, as a result of practically no other treatment than the clearing of the mouths from infection, that he wrote a scathing article which brought both professions to their feet. Among other things, Dr. Hunter said in this paper: "Sepsis in medicine therefore ranks, in my experience, as the most prevalent and potent infective disease in the body. It therefore deserves the particular attention of the whole profession as much as it has hitherto received their particular neglect. It requires this attention at the hands of every branch of the profession." Dr. Hunter's investigations seem to have been confined principally to clinical observations and case histories, yet they are of great value because of the large number and variety of cases reported.

The studies of Dr. Frank Billings and the group of men associated with him in Chicago have been along more scientific lines, and have given unquestionable proof of Dr. Hunter's observations. Dr. Billings says: "Systemic disease due to a focus of infection anywhere is probably always hematogenous. The study of infected tissues of experimentally inoculated animals, and the infected muscles, joint tissues, lymph nodes proximal to infected joints, nodes on tendons, etc., of patients, yield specific bacteria, and histologically there is found embolism of the small and terminal blood vessels. Local hemorrhage and endoarterial proliferation result in interstitial overgrowth, cartilaginous, osseous, vegetative and other morbid anatomical changes, dependent on the character of the tissue infected." This one quotation will suffice to impress the fact that Dr. Billings' investigations have been exceptionally thorough.

Dr. E. C. Rosenow has done most of the bacteriological work for Dr. Billings' cases and his investigations have demonstrated that many changes occur in the morphology of organisms in accordance with their environment, also that they develop as yet unexplained affinities for certain tissues. This was proven by the use of fresh cultures from four groups of cases; appendicitis, ulcers of the stomach, cholecystitis, and arthritis. The cultures were injected into veins of dogs and rabbits, and in each group a very large majority of the animals developed inflammations of the very same tissues as those from which the cultures were made, while there were inflammations of comparatively few of the other tissues.

In the light of our present knowledge, we are justified in making the following summary of the relation of mouth foci to general systemic conditions:

1. The mouth contains a large variety of micro-organisms, which may be divided into two groups; those which are normal or constantly present, and those occasionally or frequently found.

2. Conditions in the mouth are such that slight inflammations of the gingivae are of frequent occurrence, being present in about ninety-five per cent of mouths of adults.

3. These slight inflammations, if untreated, may gradually progress to chronic suppurations. The suppurations are caused by organisms normal to the mouth.

4. All organisms in the mouth, whether normal or accidental, have access to the blood stream through the soft granulations.

5. The normal resistance tends to prevent systemic effects and is apparently successful in the large majority of cases.

6. The transmission of infection from the primary focus is principally hematogenous.

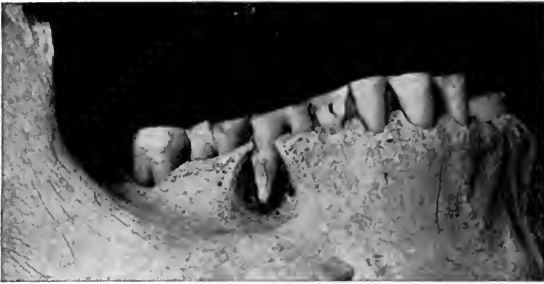


Fig. 19. Lower jaw of a Flat-Head Indian from Columbia River, Oregon, showing destruction of bone by a chronic alveolar abscess about the distal root of the first molar. The bone about the first molar of the opposite side is in practically the same condition. This is typical of the injury which occurs.

7. The primary focus is characterized by suppuration, while the secondary lesion is non-suppurative. Therefore the secondary lesion is not caused by the principal organism of the primary focus, but by other organisms which enter the primary focus with or after the pus producer, and thus gain access to the circulation; or else the morphology of the pyogenic organism is changed if it produces the secondary lesion.

8. The organisms entering the circulation through such foci appear to have an as yet unexplained tendency to locate in particular tissues.

9. The secondary effects include a very wide range of conditions. Chronic arthritis, endocarditis, nephritis, cholecystitis, ulcers of the stomach, and appendicitis are the most frequent definite lesions. General impairment of health and vigor, with or without recognizable lesions, is common.

10. The secondary effects are usually insidious in their onset and progress and, when cases present to physicians for treatment, are often difficult of management.

11. It is imperative that the primary foci be eliminated, regardless of the apparent systemic effect or lack of systemic effect.

12. For the reason that the mouth contains the primary foci in the large majority of cases, a great opportunity is open to the dental profession to prevent grave systemic disease.

The chronic foci of the mouth may be divided into three groups: (1) Deposits of salivary calculus. There may be included in this group all fillings, crowns or bridges which impinge on the soft tissues and keep them in a constant state of irritation, in somewhat the same manner as does a deposit of calculus. In all cases of this group the rule is that the systemic danger is removed with the relief of the pressure contact on the soft tissue. (2) Pus pockets alongside roots resulting from a suppurative gingivitis. (3) Chronic alveolar abscesses. Each of the latter two causes the detachment of the periodontal membrane from the cementum, and the denuded, pus-soaked cementum is the chief factor in maintaining the chronicity of these.

Today a thorough search for mouth infection is usually undertaken only in cases in which secondary symptoms are manifest, and generally after the physician has been consulted. We know that many such cases will not be benefited by the removal of the cause at this time, because the secondary effect is already too well established. May I suggest that the highest duty of the dental profession today is to search for and eliminate these foci before secondary lesions are manifest. If a minority of these foci are known to have caused serious secondary effects, the majority should be eliminated while there is the opportunity to protect the health. This is the opportunity which is before the dental profession today. Such men as Billings, Osler, Mayo, Hunter, and others have expressed it as their belief that the majority of the secondary lesions are from primary foci in the mouth, which means that the dental profession can prevent these diseases if they appreciate the situation and seize the opportunity.

In giving this course of lectures, it has been my endeavor to stimulate your interest in this subject. What I have presented is necessarily superficial and must be supplemented by an extended study of what has been written, together with careful observation and application in daily practice. If I have set some of you to thinking, so that as time passes your appreciation of these conditions will be better, I shall have accomplished my purpose.

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